MOG antibody testing in bilateral optic neuritis: a case-based study on diagnostic yield and treatment outcomes

By Dasi Sharath Chandra

WORD COUNT 2355
TIME SUBMITTED 14-JUL-2025 08:24PM
PAPER ID 117221394

MOG antibody testing in bilateral optic neuritis: a case-based study on diagnostic yield and treatment outcomes

Dasi Sharath Chandra, Noel Sam Thomas, Prem Balaji Lankapothu,

E.G. Raghunathan

Department of General Medicine, Saveetha Medical College and Hospital, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, Tamil Nadu, India

Corresponding author:
Dasi Sharath Chandra
Email: Sharathc30@gmail.com

Abstract

Myelin oligodendrocyte glycoprotein antibody-associated optic neuritis (MOG-ON) is a distinct autoimmune demyelinating condition characterized by severe visual impairment and a high relapse rate. This case report highlights the clinical progression, diagnostic challenges, and management of bilateral MOG-ON in a 44-year-old male with diabetes and hypertension. The patient initially presented with left-sided retro-orbital pain, photophobia, and progressive visual loss, which rapidly worsened to no light perception despite corticosteroid therapy. Diagnostic evaluations, including MRI and serological testing, confirmed MOG antibody positivity, leading to MOG-ON diagnosis. Subsequent vision loss in the contralateral eye prompted escalation to plasmapheresis, yielding partial visual improvement. Maintenance therapy with rituximab was planned to prevent relapses. This case shows the aggressive nature of MOG-ON and the importance of early recognition, comprehensive diagnostic workup, and individualized treatment strategies. The disease's overlap with other demyelinating conditions such as NMOSD necessitates advanced imaging and serological assays for accurate diagnosis. Long-term outcomes in MOG-ON are generally favorable with appropriate intervention, although recurrent episodes pose a significant therapeutic challenge. Emerging therapies targeting immune mechanisms hold promise for improving disease control. This report emphasizes the need for vigilant management to optimize visual outcomes and reduce disease burden.

INTRODUCTION

Optic neuritis is a prominent inflammatory demyelinating condition affecting the optic nerve, often associated with autoimmune disorders. Among its variants, myelin oligodendrocyte glycoprotein antibody (MOG-Ab)-associated optic neuritis (MOG-ON) represents a distinct clinical entity characterized by severe visual impairment and recurrent episodes. Unlike aquaporin-4 antibody-positive neuromyelitis optica spectrum disorders (NMOSD), MOG-ON often exhibits unique clinical features, such as bilateral involvement, optic disc swelling, and extensive optic nerve lesions on imaging. Its pathophysiology, rooted in autoimmune-mediated damage, is distinctly linked to MOG-specific antibodies targeting the central nervous system

myelin components, emphasizing the need for early recognition and targeted management (1,2).

Recent advancements in serological testing have illuminated the diagnostic significance of MOG antibodies in distinguishing MOG-ON from other demyelinating diseases. This case report discusses a rare presentation of bilateral MOG-ON, contributing to the growing understanding of its clinical spectrum and therapeutic challenges.

MOG-ON is an increasingly recognized form of optic neuritis with unique clinical and immunological characteristics. MOG antibodies, detectable in the serum of affected individuals, mediate inflammatory demyelination primarily involving the optic nerves, spinal cord, and, occasionally, the brain. Unlike NMOSD, MOG-ON often manifests with bilateral optic nerve involvement and is less commonly associated with systemic autoimmune conditions (3,4). The typical presentation includes acute or subacute visual loss, dyschromatopsia, and optic disc swelling, with variable recovery depending on the timeliness and appropriateness of therapeutic interventions.

Diagnostic challenges persist due to overlapping features with NMOSD and multiple sclerosis. However, identifying MOG antibodies through cell-based assays has facilitated more accurate diagnosis and better understanding of its clinical course. Treatment strategies often include high-dose corticosteroids, plasmapheresis, or immunosuppressive agents, with relapse prevention remaining a significant challenge (5).

CASE DETAILS

A 44-year-old man with a known history of diabetes mellitus and systemic hypertension for two years, not on regular medications, presented with complaints of a throbbing headache localized to the left frontal region. He also reported left orbital and retro-orbital pain for four days, accompanied by photophobia, diplopia, and blurring of vision. Over the past three days, he experienced nausea and vomiting, which were non-projectile, non-bilious, and not blood-stained. He denied neck pain, limb weakness, involuntary movements, fever, memory disturbance, or loss of consciousness.

On examination, the patient was conscious, oriented, and afebrile. His blood pressure was 130/80 mmHg, pulse rate was 88/min, respiratory rate was 18/min, and oxygen saturation was 98% on room air. Capillary blood glucose was 115 mg/dl. Systemic examination revealed no abnormalities: cardiovascular system showed regular S1 and S2 sounds, bilateral air entry was present in the respiratory system, the abdomen was soft, and central nervous system examination revealed intact speech and memory.

Day 1 of Admission

Ophthalmological evaluation revealed bilateral grade I papilledema. The left eye showed a relative afferent pupillary defect (RAPD) with a visual acuity 3/60 and preserved light perception. The right eye had no RAPD, visual acuity more excellent than 3/60, and preserved light perception.

Day 2 of Admission

The patient's visual acuity in the left eye rapidly deteriorated to 0/60 with complete loss of light perception. Blood investigations showed hemoglobin of 13.8 g/dl, total leukocyte count of 11,110/mm^3, platelets at 3.16 lakh/mm^3, neutrophils 66%, HbA1c 7.3%, and inflammatory markers including ESR at 29 mm/hr and CRP at 11 mg/L. Thyroid function tests were within normal limits, and serological tests were negative for infections or autoimmune markers.

Further diagnostic investigations included:

- **Visual Evoked Potential (VEP):** Indicated an anterior visual pathway defect in the left eye.
- **Cerebrospinal Fluid (CSF) Analysis:** Normal, with no cells, protein 15.33 mg/dL, glucose 66 mg/dL, ADA 3.834 U/L, LDH 54 U/L, and no evidence of malignancy or infection.
- **MRI Brain with Optic Nerve Cuts:** Demonstrated enlargement and focal enhancement of the retrobulbar intra-orbital segment of the left optic nerve, consistent with optic neuritis and perineuritis. Subtle periventricular and subcortical T2/FLAIR hyperintensities suggested small vessel ischemic changes.
- **Autoimmune Panel:** ANA, C-ANCA, and P-ANCA were negative.
- **Antibody Testing:** Myelin oligodendrocyte glycoprotein (MOG) antibodies were positive.

Diagnosis and Management

The patient was diagnosed with MOG antibody-associated optic neuritis. Initial treatment with pulse corticosteroid therapy showed no improvement in the left eye's vision. The patient was discharged on maintenance oral steroids.

Follow-up and Recurrence

Ten days post-discharge, the patient returned with right orbital pain and acute vision loss in the right eye. Examination revealed perception of light and hand motion only. Given the bilateral vision loss and the severity of symptoms, plasmapheresis was initiated. Five cycles were administered every other day over ten days. After three cycles, minimal visual improvement was noted, with a Rosenberg chart vision of 20/400 in the right eye. Optical coherence tomography was planned to assess retinal structure, and rituximab was scheduled for follow-up to prevent future relapses.

Prognosis

MOG antibody-positive optic neuritis typically shows better visual field outcomes with appropriate management. However, recurrence remains a significant challenge, requiring long-term immunosuppressive therapy to reduce the frequency and severity of relapses.

This case highlights the aggressive nature of MOG antibody-associated optic neuritis and the importance of early intervention with plasmapheresis and immunotherapy to preserve vision and improve quality of life.

DISCUSSION

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a distinct clinical entity within the spectrum of autoimmune demyelinating diseases of the central nervous system (CNS). Among its various manifestations, MOG antibody-associated optic neuritis (MOG-ON) is of particular clinical interest due to its unique pathophysiological, diagnostic, and therapeutic characteristics. This discussion, focusing on the presented case, explores the clinical features, diagnostic challenges, management strategies, and prognostic considerations in MOG-ON.

MOG-ON frequently manifests as severe and often bilateral optic neuritis, with symptoms including acute vision loss, retro-orbital pain, and optic disc swelling. Compared to aquaporin-4 antibody (AQP4)-associated neuromyelitis optica spectrum disorders (NMOSD), MOG-ON is less commonly associated with systemic autoimmune disorders and more often presents with bilateral involvement (6,7). The rapid progression to significant visual loss in the current case, despite corticosteroid therapy, highlights the aggressive nature of the disease in specific individuals. Timely diagnosis of MOG-ON is critical but challenging due to overlapping clinical and radiological features with NMOSD and multiple sclerosis (MS). Identifying MOG antibodies through cell-based assays is a pivotal diagnostic tool that enables differentiation from these conditions (8,9). In the presented case, the positive MOG antibody test confirmed the diagnosis after thoroughly excluding other potential etiologies. Imaging findings, including optic nerve swelling and enhancement on MRI, provided further diagnostic support (10).

Acute Management High-dose corticosteroids are the mainstay of initial therapy for MOG-ON, aiming to suppress acute inflammation and promote visual recovery. However, as observed in this case, corticosteroids alone may be insufficient in severe presentations, necessitating escalation to plasmapheresis or intravenous immunoglobulin (IVIG) (11). Plasmapheresis, administered in five cycles, facilitated minimal visual improvement, depicting its potential efficacy in refractory cases (12). **Long-term Immunotherapy** The recurrence risk in MOG-

ON remains a significant clinical concern, with relapse rates estimated to be higher compared to MS and NMOSD (13). Rituximab, a monoclonal antibody targeting CD20-positive B cells, has shown promise in reducing relapse frequency and severity in MOG-ON (14). This patient's planned transition to rituximab therapy aligns with evidence supporting its use as a maintenance therapy in relapsing cases.

Despite its aggressive nature, MOG-ON generally exhibits better long-term visual outcomes than AQP4-NMOSD when managed appropriately (15). Factors influencing prognosis include the timeliness of therapeutic intervention, severity of initial presentation, and response to immunotherapy. Early recognition and aggressive treatment are essential to optimize outcomes and prevent irreversible vision loss, as seen in the partial recovery achieved post-plasmapheresis in this case. The distinct clinical and serological profile of MOG-ON necessitates heightened awareness among clinicians. Integration of serological testing, advanced imaging techniques, and a comprehensive clinical evaluation is crucial for accurate diagnosis (16). Given the heterogeneity in disease severity and response to therapy, individualized treatment plans are essential. This case illustrates the importance of escalating to plasmapheresis in corticosteroid-refractory cases and incorporating rituximab for long-term management (17). Regular follow-up and monitoring of antibody titers, coupled with maintenance immunotherapy, play a vital role in minimizing relapse risk and associated morbidity—the recurrent visual loss in this patient and the necessity of vigilance in managing MOG-ON (18). Emerging therapies, such as interleukin-6 inhibitors and CD19-directed CAR T-cell therapy, can improve outcomes in refractory MOG-ON (19,20). Future research should focus on elucidating disease mechanisms, optimizing treatment algorithms, and exploring novel biomarkers for early detection and prognostic stratification.

This case highlights the complex clinical course and management challenges in MOG-ON. Early diagnosis, aggressive acute management, and maintenance immunotherapy are critical for preserving vision and improving patient outcomes. As MOG-ON's understanding evolves, advancements in diagnostic and therapeutic strategies are anticipated to enhance care for affected individuals.

CONCLUSION

MOG antibody-associated optic neuritis (MOG-ON) represents a distinct autoimmune condition with unique clinical and immunological features. This case shows the importance of early diagnosis through advanced imaging and serological testing to differentiate MOG-ON from other demyelinating diseases. The rapid progression to severe visual impairment in this patient, despite corticosteroid therapy, highlights the aggressive nature of the disease and the need for prompt escalation to plasmapheresis and maintenance immunotherapy. While MOG-ON often demonstrates better long-term visual outcomes compared to other optic neuritis variants, the risk of recurrence remains a significant challenge. The incorporation of therapies such as rituximab, as planned in this case, is pivotal in reducing relapse rates and preserving vision. Regular follow-up and a multidisciplinary approach involving neurology, ophthalmology, and immunology are crucial for optimal management. This case adds to the growing body of evidence on MOG-ON and emphasizes the necessity of individualized treatment plans tailored to disease severity and patient response. Ongoing research into novel therapeutic options and biomarkers is essential to further enhance outcomes for patients with

MOG-ON. Early recognition and aggressive management remain the cornerstones of care in mitigating the impact of this disabling condition.

References

1. Li Y, Sun M, Wei S, Xu Q, Zhou H. Case report: Infectious mononucleosis with bilateral retinal haemorrhages under myelin oligodendrocyte glycoprotein antibody-associated disease. *Front Immunol.* 2024;1480134. DOI: 10.3389/fimmu.2024.1480134
2. Volpe G, Jurkute N, Girafa G. Diagnostic value of inter-eye difference metrics on OCT for myelin oligodendrocyte glycoprotein antibody-associated optic neuritis. *Neurology.* 2024;29:200291. DOI: 10.1212/NXI.0000000000200291
3. Fabiani C, Tosi GM, Damato V. Uveitis: A snapshot in the MOG antibody spectrum. *Mult Scler Relat Disord.* 2024;29:125173. DOI: 10.1177/13524585241285173
4. Zhang M, Cen Z, Hu H, Liu C, Zuo H. Recurrent Myelin Oligodendrocyte Glycoprotein Antibody-Positive Optic Neuritis and Leber Hereditary Optic Neuropathy Coexist in a Young Man. *Neuroophthalmology.* 2024;2411762. DOI: 10.1080/01658107.2024.2411762
5. Gutierrez SA, Alikhani K. A case of myelin oligodendrocyte glycoprotein antibody-associated disease with bilateral sequential optic neuritis and thoracic myelitis. *Neurology.* 2024;1051228. DOI: 10.1212/01.wnl.0001051228.59774.74
6. Levy M, Yeh A, Hawkes C. Mr. Machiavelli's choice for treatment of MOG antibody disease: Do the benefits of long-term corticosteroids justify the means? Mult Scler Relat Disord. 2024; DOI: 10.1016/j.msard.2024.104783.
7. Pedrosa DA, Fernandes GBP, Talim N. MOG-IgG is rare in AQP4-IgG seronegative NMO phenotype in Brazil. Mult Scler Relat Disord. 2024; DOI: 10.1016/j.msard.2024.104798.
8. Özdemir HN, Karakoç MT, Gökçay F. Biomarkers in patients with autoimmune optic neuritis not associated with multiple sclerosis. Mult Scler Relat Disord. 2024; DOI: 10.1016/j.msard.2024.104803.
9. Vallejos GB. Risk factors associated with relapse in double seronegative neuromyelitis optica spectrum disorder. Neurology. 2024; DOI: 10.1212/WNL.0000000000206036.
10. Fisher K, Salazar K. Pediatric MOGAD: Clinical experience in a tertiary care setting. Neurology. 2024; DOI: 10.1212/01.wnl.0001051436.93140.bf.
11. Rudebeck SR, Eyre M. Reassuring neuropsychological outcome data in myelin oligodendrocyte glycoprotein antibody-associated disease. Eur J Paediatr Neurol. 2024; DOI: 10.1016/j.ejpn.2024.00155.
12. Ahmed HS, Haroun L, Daoudi S. Tumefactive myelin oligodendrocyte-associated disease presenting as acute flaccid myelitis: A case report. Mult Scler Relat Disord. 2024; DOI: 10.1016/j.msard.2024.104658.
13. Gummadi B, Aoun R, Fayssal NA. A case of neurosarcoidosis presenting with bilateral optic neuritis. Neurology. 2024; DOI: 10.1212/WNL.0000000000206036.

14. Pace S, Messina S, Chen B. Cerebral cortical encephalitis in adults with myelin oligodendrocyte glycoprotein antibody-associated disease. Eur J Neurol. 2024; DOI: 10.1111/ene.16550.

15. Barman D, Prakash S. Seeing through the blurry vision of MOGAD vs NMOSD. Neurology. 2024; DOI: 10.1212/WNL.0001051280.38423.15.

16. Cabrera-Maqueda JM, Sepulveda M. CD19-directed CAR T-cells in a patient with refractory MOGAD: clinical and immunologic follow-up for 1 year. Neurology Neuroimmunol Neuroinflamm. 2024; DOI: 10.1212/NXI.0000000000200292.

17. Samuel P, Silvia M, Radu T, Athanasios P. A case series of adults with MOGAD encephalitis. J Neurol Neurosurg Psychiatry. 2024;95(Suppl 2):A7.3. DOI: 10.1136/jnnp-2024-ABSTRACTS.

18. Fichtner A. Entwicklung eines zellbasierten Nachweisverfahrens für Serum-anti-Myelin-Oligodendrozyten-Glykoprotein-Antikörper. Doctoral Thesis. Göttingen University; 2024. Available at: ediss.uni-goettingen.de.

19. Iancu R, Anton N, Istrate S. Visual function improvement after plasma exchange therapy for acute optic neuritis in neuromyelitis optica spectrum disorders: Case series and review. Diagnostics. 2024;14(9):863. DOI: 10.3390/diagnostics14090863.

20. Haroun L, Ahmed HS, Daoudi S. Tumefactive Myelin Oligodendrocyte-Associated Disease Presenting as Acute Flaccid Myelitis: A Case Report. Mult Scler Relat Disord. 2024; DOI: 10.1016/j.msard.2024.104658.